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9/3/08

J. Douglas Bremner, M.D.
Report for Michael Passino
RE: Jon Hall

I. Introduction

Terms of Engagement and Purpose: Michael Passino and Paul Bottei asked me to review certain psychological evaluations of Mr. Jon Hall, including a neuropsychological report by Dr. Ruben Gur, an overview of Mr. Hall's personal history, and the facts and circumstances surrounding the death of Mr. Hall's wife Billie Jo, to determine what effect, if any, Mr. Hall's exposure to childhood neglect and abuse had on the development of his brain, and in turn, what if any effect this had on Mr. Hall's ability to commit first-degree premeditated murder as defined by the State of Tennessee.

In setting out my opinions and conclusions, Section II identifies the documents and information I reviewed; Section III outlines my qualifications; Section IV provides a brief client history; Section V describes generally the effects of traumatic stress, and its effects on cognitive function and brain structure and function; and Section VI provides a summary of my conclusions, explaining the effects of traumatic stress on Jon Hall and the effect of such changes in Mr Hall's brain structure and function, to a reasonable degree of medical certainty, on his conduct, perceptions and actions at the time of his wife's death, Billie Jo Hall.

II. Documents/information reviewed

- Summary of 2002 testing of Dr. Pamela Auble
- 2/27/08 letter of Dr. Ruben Gur, analyzing enclosed neurocognitive, MRI and PET results
- A social history of Mr. Hall
- Transcripts of Mr Hall's trial
- Jury instructions to Mr Hall's jury on elements of first-degree premeditated murder

- In the course of arriving at my opinions and conclusions I also had brief telephone conversations with Messrs. Bottei and Passino during the course of this engagement
- These are, of course, the kinds and types of information upon which I and others in my profession customarily rely in forming a medical conclusion or opinion

III. Qualifications

I am currently a tenured Professor of Psychiatry and Behavioral Sciences at Emory University School of Medicine. Additionally, I hold a joint and secondary appointment as Professor of Radiology at the Emory University School of Medicine. I am Director of the mental health research facilities at the Atlanta Veterans Administration Hospital, and the director of the Emory Clinical Neuroscience Research Unit.

I have been board certified in psychiatry and neurology since 1998 and have been board certified in nuclear medicine since 2001.

In 1983, I received a Bachelor of Arts degree with Honors from the University of Puget Sound in Tacoma, Washington. In 1987, I received my medical degree from Duke University School of Medicine in Durham, North Carolina. Following that, I was a resident in Psychiatry at Yale University School of Medicine in New Haven, Connecticut from 1987-1991. Additionally, I underwent a residency in Nuclear Medicine at the Department of Diagnostic Radiology at Yale University School of Medicine. I was Medical Director of the Inpatient Unit of the National Center for PTSD at West Haven VA Medical Center 1991-1992. I was also an assistant professor in the Department of Psychiatry at Yale University School of Medicine from 1992 to 1995, and was an Assistant Professor and then Associate Professor of Diagnostic Radiology and Psychiatry at the Yale University School of Medicine 1997-2000. I was the Director of the Emory Center for Positron Emission Tomography from 2000-2006.

I am a member of the Society for Nuclear Medicine, the Society of Biological Psychiatry, the Society for Neuroscience and a member of The American College of Neuropsychopharmacology.

I am a member of a number of scientific review committees including the National Institute of Health Center for Scientific Review (CSR) Bio-behavioral Mechanisms of Emotion, Stress and Health (MESH) and the Veterans Administration Mental Health and Behavioral Sciences Merit Review Committee. Additionally, I have served on the editorial board of *Biological Psychiatry*, *Neuropsychopharmacology*, *Psychopharmacology Bulletin* and *Journal of Trauma and Dissociation*. I have also served as a manuscript reviewer for a number of professional journals, as set forth in my CV.

I have written over 200 journal articles and book chapters, primarily on the subject of posttraumatic stress disorder (PTSD), edited two books, including (co-editor with C. Marmar) *Trauma, Memory and Dissociation*, APA Press, Washington DC, 1998; (co-editor with PA Saigh) *Posttraumatic Stress Disorder: A Comprehensive Text*. Allyn & Bacon, Needham Heights, MA: 1999; and authored three books: *Does Stress Damage the Brain? Understanding Trauma-related Disorders from a Mind-Body Perspective*. New York: W. W. Norton, 2002; *Brain Imaging Handbook*. New York: W. W. Norton,

2005; *Before You Take that Pill: Why The Drug Industry May be Bad for Your Health: Risks and Side Effects You Won't Find on the Label of the Most Commonly Prescribed Drugs, Vitamins and Supplements*. New York: Avery, 2008.

I have twenty years of experience as a psychiatrist specializing in PTSD and currently perform assessments on returning Iraq veterans with PTSD. I have been funded for 18 years by various sources including NIH and VA for research in the neurobiology of PTSD and depression.

A complete CV is attached hereto as an Exhibit, which lists all publications authored by me and set forth additional details regarding my qualifications to express my opinions in this case.

IV. Client History

It is apparent that Mr Hall was exposed to extreme violence and neglect as a child. As discussed below, and as the neuropsychological testing and report of Dr. Ruben Gur demonstrate, exposure to traumatic stress has neurobiological and cognitive effects, changing the development, structure and function of the brain, but also a person's behavior and how a person views and responds to the world around them.

The client is a 43 year old white male who was convicted of first degree murder in 1997 in Jackson, Tennessee, in the death of his wife Billie Jo on July 29, 1994. Appeal in the Tennessee courts upheld the conviction of first-degree murder and sentence of death. His case is currently pending in federal district court in a habeas corpus proceeding. At issue is whether the client's actions were premeditated. For a number of reasons which I will set forth below it is my opinion that the actions of the client were not premeditated. In addition there are several mitigating circumstances which impacted on his case.

Mr Hall was born August 5, 1964, and grew up in Ligonier, Pennsylvania, and was the youngest of seven children to Carol Hood Hall Alexander (Carol Alexander) and Jay Hall. Mr Hall's mother married his father when she was 15 years old. Jay Hall's father was a violent and abusive alcoholic who beat his wife, chased her with a gun, and once knocked Jon's sister off a chair and broke her collarbone. Jon Hall was born the last of seven children. He had jaundice in infancy, had his umbilical cord wrapped around his neck, and was seven weeks premature. Testimony of his siblings and his mother showed that he was the victim of substantial abuse and neglect in childhood. His father was an alcoholic who beat his wife often. They were engaged in what was described by one sister as "constant" arguing. Typically the father would come home late at night intoxicated and the parents would start arguing which often led to physical blows. One fight was described where the father pulled handfuls of hair off the head of their mother and beat her so severely that blood came out of her ears and nose. The children would try and keep sharp objects hidden because they were afraid that their parents would kill each other. His father claimed that Jon was not his biological son and treated him differently from the other children, e.g. he wouldn't pick up Jon and would buy ice cream for his brothers and have them go home and eat it in front of him just so he could see what he was missing. He was physically abused by older siblings and their friends and received no protection from his father. His father beat him with belts that caused welts and hit him with a knife inside of a sheath. The girls in the family were sexually abused by the older boys. Jon's sister Sheryl said that "mom totally ignored us kids... we were an

inconvenience, that is all we ever were. I have abandonment issues.” Because of poverty the children were raised at times in different homes with their grandparents. Jon’s father died of a heart attack in 1974. His mother remarried and he continued to receive emotional abuse from his step-father.

An older sister of Jon had him move into their house in North Carolina in his last year of high school because she was concerned about him living with his mother and step-father. He got a job as a mechanic and met Billie Joe. They were married on May 14, 1988. She had two daughters from a prior relationship and they had two more daughters in 1988 and 1991, one of whom had cerebral palsy (CP) with severe handicaps. He was described by his sisters and mother as very affectionate with all of the girls and was the primary caretaker for his disabled daughter while his wife worked and went to school. He drank heavily and was described as alcohol dependent by a psychologist who saw him prior to his trial and had more than one driving under the influence (DUI) convictions but other than that had no criminal record. In spite of this his employer described him as hard working. He was always on time and reliable and worked on Saturdays. Some days he would bring his children with him and take care of them while he worked. Several months before the murder his relationship with his wife got worse and he moved out. During this time his employer said that his work performance got worse and he often talked about how he felt depressed about his wife. He then quit his job which his employer described as the result of him acknowledging that he was no longer able to adequately perform his job.

In July 1994 his wife got a protective order against him and filed suit for divorce. Just before July 29, 1994, Jon Hall’s sister Sheryl and brother Jeff were trying to get emergency psychiatric treatment for him, as they perceived that he was extremely distraught about the possibility of losing his children and wife.

On the day of the murder he had been drinking and communicated with his wife about going over to the house later to bring her some money. The fact that he had been drinking was an additional factor, added on to his abnormal brain function, that would contribute to his inability to properly regulate emotion, as well as to think properly and act in a logical and deliberate manner. A money order for \$25.00 made out to her was later found at the house. That evening he went over to his wife’s house where she was watching television with their daughters. According to the statements given to the police by the daughters he initially entered the house in a calm fashion and did not initially argue with his wife. They reported that he brought some beer with him and took a beer out and started to drink it. They later began arguing and he began hitting her and then dragged her outside to a pool, pushed her into the pool and then left in her van. He later stated that he was unaware that she died in the pool. The cause of death was ruled as asphyxiation, which could have been caused by drowning in the pool. After leaving the scene he drove to his brother’s house in Texas where he was arrested without incident. When he was arrested he stated that he was not aware that she had died.

In prison he was seen by a psychologist (Dr Joe Mount) for problems with depression and he expressed great remorse and sadness about the death of his wife and concern about the effect on his children. He was placed on suicide watch in September and October of 1994. He received the diagnosis of adjustment disorder with mixed emotional features (depression and anxiety) and substance abuse and dependency (alcohol). Post conviction

(2002) he was evaluated by Keith Caruso MD and diagnosed with intermittent explosive disorder, major depression, alcohol and cannabis dependence, narcissistic and borderline personality disorders, and antisocial personality disorder.

Mr Hall was evaluated on Feb 17, 2008, by Dr. Rachel Gur of the University of Pennsylvania, an internationally recognized expert in the field of brain imaging. Dr. Gur performed positron emission tomography (PET) and magnetic resonance imaging (MRI) brain imaging and neuropsychological testing of Mr Hall and identified metabolic abnormalities in the hippocampus, amygdala, and corpus callosum, all brain areas identified as being abnormal in individuals with a history of early childhood trauma. In addition he had smaller volume of the frontal cortex, a brain area that plays an important role in emotional regulation.

V. Traumatic Stress and Its Neurobiology

A. General Effects of Traumatic Stress

Traumatic stress, including childhood neglect and abuse, can have a range of effects on mental conditions, including posttraumatic stress disorder (PTSD), depression, borderline personality disorder, and alcohol and substance abuse and dependence. Posttraumatic stress disorder (PTSD) is recognized as a distinct psychiatric disorder in the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).¹ PTSD is defined as a co-occurrence of typical symptoms, or 'diagnostic criteria.' Descriptive studies have established the validity of disorder's symptom structure, evaluated its natural course and described consistent associations between the PTSD and psycho-physiological, neuroendocrine and brain imaging findings. The disorder is diagnosed by ascertaining the presence of qualifying diagnostic criteria on the basis of the patient's report, clinical observation and corroborating information. The effects of traumatic stress have lasting effects on the brain which are described below; this affects the capacity for reason, correct thinking, and self control of behavior.

B. Neurobiology of Traumatic Stress

Traumatic stress induced mental disorders represent the behavioral manifestation of stress-induced changes in brain structure and function.² Stress results in acute and chronic changes in neurochemical systems and specific brain regions, which result in long-term changes in brain "circuits", involved in the stress response.²⁻⁵ Brain regions that play an important role in trauma related mental disorders include hippocampus, amygdala, and medial prefrontal cortex. Cortisol and norepinephrine are two neurochemical systems that are critical in the stress response.

The corticotropin releasing factor (CRF)/hypothalamic-pituitary-adrenal (HPA) axis system plays an important role in the stress response. Corticotropin-releasing factor (CRF) is released from the hypothalamus, with stimulation of adrenocorticotropin hormone (ACTH) release from the pituitary, resulting in glucocorticoid (cortisol in man) release from the adrenal, which in turn have a negative feedback effect on the axis at the level of the pituitary as well as central brain sites including hypothalamus and

hippocampus. Cortisol has a number of effects which facilitate survival. In addition to its role in triggering the HPA axis, CRF acts centrally to mediate fear-related behaviors,⁶ and triggers other neurochemical responses to stress such as the noradrenergic system via the brainstem locus coeruleus.⁷ Stress also results in activation of the noradrenergic system, centered in the locus coeruleus. Noradrenergic neurons release transmitter throughout the brain which is associated with an increase in alerting and vigilance behaviors, critical for coping with acute threat.⁸⁻¹⁰

Studies in animals showed that stress has lasting effects on the HPA axis and norepinephrine. Stressed animals demonstrated an inability to terminate the glucocorticoid response to stress,^{11, 12} as well as deficits in fast-feedback of glucocorticoids on the HPA axis.¹³ These effects are mediated by an increase in synthesis of CRH mRNA following stress.¹⁴ In nonhuman primates, adverse early experiences resulted in long-term effects on behaviors, as well as elevated levels of corticotropin releasing factor (CRF) in the cerebrospinal fluid.¹⁵ Exposure to chronic stress results in potentiation of noradrenergic responsiveness to subsequent stressors and increased release of norepinephrine in the hippocampus and other brain regions.¹⁰

Preclinical and clinical studies have shown alterations in memory function following traumatic stress¹⁶ as well as changes in a circuit of brain areas, including hippocampus, amygdala, and medial prefrontal cortex, that mediate alterations in memory and control emotions, thinking and behavior.¹⁷ The hippocampus, a brain area involved in verbal declarative memory, is very sensitive to the effects of stress. Stress in animals was associated with damage to neurons in the CA3 region of the hippocampus (which may be mediated by hypercortisolemia, decreased brain derived neurotrophic factor, and/or elevated glutamate levels) and inhibition of neurogenesis.¹⁸⁻²³ High levels of glucocorticoids seen with stress were also associated with deficits in new learning.^{24, 25}

Long-term dysregulation of the HPA axis is associated with PTSD, with low levels of cortisol found in chronic PTSD²⁶⁻³¹ and elevations in CRF^{27, 32} Exposure to a traumatic reminder is associated with a potentiated release of cortisol in PTSD.³³

In summary, traumatic stress has lasting effects on brain regions including the hippocampus, amygdala and medial prefrontal cortex, as well as neurochemical systems including cortisol and norepinephrine. These changes also result in changes in behavior, so that a traumatized person with PTSD will not have the same ability to control behaviors, emotions or thinking as a normal person would. Based on this Jon Hall, with his history of early abuse and neglect, and the documented alterations in his brain, would be less likely than normal persons to act in a deliberate fashion, with a cool purpose, or to exercise normal reflection and judgment.

C. Cognitive Function and Brain Structure in PTSD

Studies in PTSD are consistent with changes in cognition and brain structure. Multiple studies have demonstrated verbal declarative memory deficits in PTSD.^{16, 34-36} Patients with PTSD secondary to combat³⁷⁻⁴¹ and childhood abuse^{42, 43} were found to have deficits in verbal declarative memory function based on neuropsychological testing. Studies, using a variety of measures (including the Wechsler Memory Scale, the visual and verbal components of the Selective Reminding Test, the Auditory Verbal Learning Test, Paired

Associate Recall, the California Verbal New Learning Test, and the Rivermead Behavioral Memory Test), found specific deficits in verbal declarative memory function, with a relative sparing of visual memory and IQ.^{37-41, 43-52} These studies have been conducted in both patients with PTSD related to Vietnam combat,^{37-41, 44, 47-49, 51} rape,⁴⁵ the Holocaust,⁵²⁻⁵⁴ adults with early childhood abuse,⁴³ and traumatized children.⁴⁶ One study in adult rape survivors showed that verbal declarative memory deficits are specifically associated with PTSD, and are not a non-specific effect of trauma exposure.⁴⁵ Other types of memory disturbances studies in PTSD include gaps in memory for everyday events (dissociative amnesia),⁵⁵ deficits in autobiographical memory,⁵⁶ an attentional bias for trauma-related material,⁵⁷⁻⁶⁶ and frontal lobe-related impairments.⁶⁷ These studies show that trauma with associated PTSD results in deficits in memory, which will affect the ability of the traumatized person with PTSD to think in a deliberate and logical fashion.

These effects were specific to verbal (not visual) memory, and were significant after controlling for IQ. Some of these studies used neuropsychological tests of declarative memory, such as Wechsler Memory Scale (WMS) and Selective Reminding Test (SRT), that have been validated as sensitive to loss of neurons in the CA3 region of the hippocampus in epileptics who underwent hippocampal resection.^{68, 69} Vietnam veterans with PTSD had 8% smaller right hippocampal volume based on MRI relative to controls matched for a variety of factors such as alcohol abuse and education ($p < .05$); smaller volume was correlated with deficits in verbal declarative memory function as measured with the WMS.⁷⁰ Another study showed a 12% reduction in left hippocampal volume in 17 patients with childhood abuse-related PTSD compared to 17 case-matched controls that was significant after controlling for confounding factors.⁷¹ Smaller hippocampal volume was shown to be specific to PTSD within the anxiety disorders, and was not seen in panic disorder.⁷² Gurvits et al.⁷³ showed bilateral hippocampal volume reductions in combat-related PTSD compared to combat veterans without PTSD and normal controls. Combat severity was correlated with volume reduction. Stein et al.⁷⁴ found a 5% reduction in left hippocampal volume in women with early childhood abuse. Other studies in PTSD have found smaller hippocampal volume and/or reductions in NAA, a marker of neuronal integrity.⁷⁵⁻⁷⁹ In a recent meta-analysis we pooled data from all of the published studies and found smaller hippocampal volume for both the left and the right sides, equally in adult men and women with chronic PTSD, and no change in children.⁸⁰ Two independent studies have shown that PTSD patients have deficits in hippocampal activation while performing a verbal declarative memory task.^{75, 81} Stress-induced hippocampal dysfunction likely mediates many of the symptoms of trauma related mental disorders like PTSD and which are related to memory dysregulation, including both explicit memory deficits as well as fragmentation of memory in abuse survivors. These effects on the brain lead to a diminished capacity for logical thinking and deliberate activity. In addition the hippocampus is involved in the control of emotion, and damage to this area will result in an inability to control emotions and act in a logical and deliberate way.

In addition to the hippocampus, other brain structures are involved in a neural circuitry of stress including the amygdala and prefrontal cortex. The amygdala is involved in memory for the emotional valence of events, and plays a critical role in the acquisition of fear responses.⁹⁴⁻⁹⁶ The medial prefrontal cortex includes the anterior cingulate gyrus (Brodmann's area 32) and subcallosal gyrus (area 25) as well as orbitofrontal cortex.

Studies demonstrated that the medial prefrontal cortex modulates emotional responsiveness through inhibition of amygdala function.⁹⁷⁻⁹⁹ Conditioned fear responses are extinguished following repeated exposure to the conditioned stimulus (in the absence of the unconditioned (aversive, e.g. electric shock) stimulus. This inhibition is mediated by medial prefrontal cortical inhibition of amygdala responsiveness.

Animal studies also show that early stress is associated with a decrease in branching of neurons in the medial prefrontal cortex.¹⁰⁰ Rauch and colleagues found smaller volume of the anterior cingulate based on MRI measurements in PTSD;¹⁰¹ another study replicated these findings in women with abuse and PTSD.⁸⁰ Other studies in abused children^{102, 103} and adults abused as children with PTSD show smaller volume of the corpus callosum. In summary, studies in both animals and humans have shown that traumatic stress has effects on the same brain regions specifically shown to be abnormal in Jon Hall, and that changes in these brain regions affect the capacity for logical thought, the ability to act in a deliberate way, and the ability to control emotions.

D. Neural Circuits in Traumatic Stress

Brain imaging studies have shown alterations in a circuit including medial prefrontal cortex (including anterior cingulate), hippocampus and amygdala in patients with trauma-related mental disorders. Many of these studies have used different methods to trigger symptoms (e.g., using traumatic cues) and then look at brain function. Stimulation of the noradrenergic system with yohimbine resulted in a failure of activation in dorsolateral prefrontal, temporal, parietal and orbitofrontal cortex, and decreased function in the hippocampus.¹⁰⁴ Exposure to traumatic reminders in the form of traumatic slides and/or sounds or traumatic scripts was associated with an increase in PTSD symptoms, decreased blood flow and/or failure of activation in the medial prefrontal cortex/anterior cingulate, including Brodmann's area 25, or subcallosal gyrus, area 32 and 24, as measured with PET or fMRI.¹⁰⁵⁻¹¹⁴ Other findings in studies of traumatic reminder exposure include decreased function in hippocampus,¹⁰⁷ visual association cortex,^{107, 111} parietal cortex,^{107, 110, 111, 115} and inferior frontal gyrus,^{107, 110, 111, 115} and increased function in amygdala,^{112, 115} posterior cingulate,^{105, 107, 108, 111} and parahippocampal gyrus.^{105, 107, 109} Shin and colleagues found a correlation between increased amygdala function and decreased medial prefrontal function with traumatic reminders,¹¹² indicating a failure of inhibition of the amygdala by the medial prefrontal cortex could account for increased PTSD symptoms with traumatic reminders. Other studies found increased amygdala and parahippocampal function and decreased medial prefrontal function during performance of an attention task,¹¹³ increased posterior cingulate and parahippocampal gyrus and decreased medial prefrontal and dorsolateral prefrontal during an emotional Stroop paradigm,¹¹⁶ and increased amygdala function with exposure to masked fearful faces.¹¹⁷ Retrieval of emotionally valenced words¹¹⁸ (e.g. "rape-mutilate") in women with PTSD from early abuse resulted in decreases in blood flow in an extensive area which included orbitofrontal cortex, anterior cingulate, and medial prefrontal cortex (Brodmann's areas 25, 32, 9), left hippocampus, and fusiform gyrus/inferior temporal gyrus, with increased activation in posterior cingulate, left inferior parietal cortex, left middle frontal gyrus, and visual association and motor cortex.¹¹⁹ Another study found a failure of medial prefrontal cortical/anterior cingulate activation, and decreased visual association and parietal cortex


function, in women with abuse and PTSD relative to women with abuse without PTSD, during performance of the emotional Stroop task (i.e. naming the color of a word such as "rape").¹²⁰ One study found increased amygdala activation with classical fear conditioning (pairing a shock and a visual stimulus), and decreased medial prefrontal cortex function with extinction, in abuse-related PTSD.¹²¹ The findings described above point to a network of related regions mediating symptoms of PTSD, including medial prefrontal cortex, hippocampus, and amygdala,¹²² brain areas that play a critical role in the regulation of thinking, control of emotion, and that determine the ability of individuals to act in a cool, rationale and deliberate fashion.

In summary, traumatic stress has lasting effects on brain regions including the hippocampus, amygdala and medial prefrontal cortex, as well as neurochemical systems including cortisol and norepinephrine. These changes also result in changes in behavior, so that a traumatized person with PTSD will not have the same ability to control behaviors, emotions or thinking as a normal person would. Based on this Jon Hall, with his history of early abuse and neglect, and the documented alterations in his brain, would be less likely than normal persons to act in a deliberate fashion, with a cool purpose, or to exercise normal reflection and judgment.

IV. Summary: The Effects of Traumatic Stress on Jon Hall

It is my opinion that in the case of Jon Hall there are a number of factors that go against the conclusion that he committed murder in the first-degree, i.e. coolly planned, deliberate, intentional, and with cool purpose, and after reflection, judgment and planning. Brain and neuropsychological testing by Dr Gur show abnormalities in brain areas involved in the regulation of thinking, emotion and behavior, which would impair his ability to think in a cool and deliberate fashion, and to adequately use reflection and judgment to plan and execute a pre-meditated murder. Severe childhood abuse and neglect affected his neurodevelopment, leading to changes in brain regions involved in mood and emotion including frontal cortex, amygdala, hippocampus and corpus callosum. These findings are consistent with a wide range of evidence from both animal and human studies of the effects of traumatic stress and early neglect on the brain, showing that trauma affects brain areas involved in regulation of emotion, thinking, and deliberate planning. The evidence presented is not consistent with a planned, intentional and calculated pattern of behavior involving a premeditated plan to kill his wife. For instance, children's statements that there was a period of calm discussion before violent argument, that he brought a money order for his wife, that he wanted to be reconciled with his wife, etc., are not consistent with coolly planned, intentional murder executed after calm reflection. He also had strong affections for his children that would not lead him to purposively kill their mother. These findings, in addition to the circumstances related to abnormalities of the brain, and the effect of alcohol intoxication, show that he did not act in a deliberate, coolly planned, and intentional way, or after planning and reflection, in order to kill his wife. Early trauma has been associated with changes in function and structure of brain areas including frontal cortex, amygdala and hippocampus, that were shown to be abnormal in Mr Hall and that play an important role in violence, emotion, aggression and behavioral control.

Sincerely,



J. Douglas Bremner, M.D.

Professor of Psychiatry and Radiology &

Emory University School of Medicine, Atlanta, Georgia;

Director, Mental Health Research, Atlanta VAMC, Decatur, Georgia

Attachments and Appendices

Exhibit 1: CV

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